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Lamellar Macular Hole: State of the Art

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Keywords
Lamellar macular hole · Epiretinal membrane · Internal limiting membrane · Müller cells

Abstract
Lamellar macular hole (LMH) is a vitreoretinal disorder characterized by an irregular foveal contour, a break in the inner fovea, dehiscence of the inner foveal retina from the outer retina, and the absence of a full-thickness foveal defect with intact foveal photoreceptors. The pathogenesis is only partially known. The advent of high-resolution optical coherence tomography has allowed distinguishing between two types of epiretinal membrane (ERM) associated with LMH: a conventional ERM (commonly found in macular pucker) and an atypical ERM (known by varied names: dense, epiretinal proliferation, or degenerative). These two types of ERM not only influence LMH morphology but also differ in cell and collagen composition. It remains unclear if these two types are indeed two distinct clinical entities or rather two stages of the same macular disorder. Studies of the natural evolution of LMH have not fully resolved this issue and also offered variable results. Surgical treatment leads to excellent anatomical and functional outcomes, but not without risks. This review provides a critical summary of the available data on LMH including some new insights.

Introduction

The term lamellar macular hole (LMH) was originally introduced by J.D. Gass in 1975 [1]. Gass reported a case of LMH secondary to cystoid macular edema after intracapsular cataract surgery. Ophthalmoscopic observation revealed a well-defined oval shape of a foveal lesion characterized by a foveal reflex, suggesting the presence of a residual retinal layer. The retinal surface in the macular area had a cellophane-like appearance. The lesion was interpreted as an LMH and the postmortem histologic study confirmed the diagnosis of no full-thickness macular hole (FTMH). The advent of optical coherence tomography (OCT) allowed the observers to describe the morphological characteristics of the LMH in more detail [1].

Witkin et al. [2] were the first to define the diagnostic criteria for LMH: an irregular foveal contour, a break in the inner fovea, dehiscence of the inner foveal retina from the outer retina, and the absence of a full-thickness foveal defect with intact foveal photoreceptors. These criteria clearly defined LMH, distinguishing it from other vitreoretinal disorders such as FTMH and macular pseudohole. However, the evolution of OCT and new studies highlighted the limitations of these criteria: LMH is not always characterized by an intact photoreceptor layer, and it is often associated with an epiretinal membrane (ERM) and intraretinal cysts (IRCs) [3–10]. Moreover,

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immunohistochemical and microscopic studies have shown differences in cell composition of ERMs, identifying two types of ERM. This fact raised the question as to whether there are two distinct clinical entities or two different stages of the same disorder [3, 7–9, 12, 13]. The different names adopted for LMH and the associated ERMs are the result of disagreement about the final definition of this disorder [2, 3, 5–9, 14]. The purpose of this paper is to review and update current information about LMHs.

This study followed the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines [13]. A total of 247 articles were collected and 84 were included in the review. RevMan 5.3 (The Cochrane Collaboration, Oxford, UK) was used to perform the meta-analysis of the collected data [15].

Epidemiology

Nine articles were analyzed with regard to the epidemiology of LMH. Five studies were excluded because they were based on clinical evaluations or ocular fundus retinography, which have a lower sensitivity than OCT [16–24]. Only 4 studies used OCT [16–19]. The Handan Eye Study [21] did not investigate the prevalence of LMH but only of ERM. Pang et al. [22] retrospectively analyzed a subpopulation of patients affected by different macular diseases (LMH, FTMH, macular pseudohole, and macular pucker), and data were collected from the database of the spectral-domain OCT (SD-OCT) archive of a vitreoretinal referral practice unit. Therefore, the included subjects were not representative of the general population.

The other 2 trials, the Maastricht [23] and Beaver Dam Eye [24] studies, were population-based cohort studies and satisfied the criteria for recruitment to a random sample. The Maastricht study [23], a large observational prospective cohort study, investigated the etiology, complications, and comorbidities of type 2 diabetes mellitus, focusing on the phenotypic aspect of the disease. The study included 3,451 participants aged 40–75 years who lived in the southern part of the Netherlands. OCT was performed on 2,660 of them. In brief, this study also focused on the prevalence of vitreoretinal interface disorders according to age, gender, and glucose metabolism status. The statistical analysis of the data revealed a prevalence of LMH of 1.1% (17 of 1,531 patients) in the subpopulation with normal glucose metabolism, of 0.7% (5 of 401 patients) in the subpopulation with pre-diabetes, and of 0.3% (2 of 728 patients) in the subpopulation affected by type 2 diabetes mellitus. The prevalence of LMH showed no significant correlation with age [23].

The second study, the Beaver Dam Eye study [24], evaluated the prevalence of vitreoretinal interface disorders (ERM, vitreomacular traction, FTMH, LMH, and macular and paravascular cysts) among 1,910 participants who underwent OCT. LMH was present in 64 eyes of 56 participants (3.6%; bilateral in 17.9%). The prevalence of LMH varied with age from 2.1% among those aged between 63 and 74 years to 2.6% among those over 85 years, without statistically significant differences. No correlation with gender was found ($p = 0.70$) [24]. LMH mainly affects subjects within the age range of 50–70 years [21–24]. According to these 2 studies [23, 24], the prevalence of LMH in the general population ranges from 1.1 to 3.6%, with no significant correlation with gender and age.

Pathogenesis

LMH is idiopathic in most cases, but it may be secondary to: cystoid macular edema due to metabolic or vascular retinal diseases [25–29], retinochoroiditis [30], age-related macular degeneration [31], retinitis pigmentosa [32], X-linked retinoschisis [33], myotonic dystrophy [34], Alport syndrome [35], Coats' disease [36], high myopia [10, 37–40], ocular trauma [41, 42], or iatrogenic damage during intravitreal injection or surgery [43–48].

After Gass' theory [1], which holds that LMH is due to spontaneous dehiscence of an internal wall of an IRC, more attention has been paid to the role of the vitreous in LMH pathogenesis, leading one to believe that LMH represents the result of an abortive process of FTMH formation. Anterior-posterior traction forces, exerted by the posterior hyaloid on the fovea due to partial posterior vitreous detachment (PVD), may cause the development of IRCs, and following complete PVD, the inner wall of an IRC may be detached, causing the formation of an LMH [49, 50]. The presence of an operculum in the vitreous in front of the fovea, adherent to the posterior hyaloid, confirms a detachment of the inner wall of an IRC. SD-OCT improved the diagnostic evaluation of the vitreoretinal interface. Following SD-OCT analysis, the detection of ERMs associated with LMHs increased from 62% [2] to 89% [50], and more recently it has been detected in all cases of LMH [3, 5, 6, 8, 9, 11, 50].

New studies have highlighted further morphological details that allowed researchers to define the distinctive features of LMHs and of associated ERMs more accurate-

ly. Since 2006, many studies [3, 5, 6, 8, 9, 11, 51] have distinguished two types of ERM associated with LMH. The first type, commonly found in macular pucker, is characterized tomographically by a hyperreflective line just above the nerve fiber layer, associated with tractional signs such as IRCs and stretching of the intraretinal layers. The second type is characterized by a band with reflective content, not associated with IRCs and tractional signs but rather with tissue defects similar to intraretinal cavitation. This second type is known by different names: dense, epiretinal proliferation, and degenerative [3, 5, 6, 8, 9, 11, 51]; in our paper it will be named "atypical ERM" (A ERM), whereas the first type will be named "conventional ERM" (C ERM).

It is not yet clear whether these are two different types of ERM or rather two different stages of the same pathological disorder. It has been shown that in some LMHs, the ERM shows characteristics of both types [6, 9–11] (Fig. 1). However, LMH development cannot be explained only by morphological characteristics. Knowledge of the histologic composition of implicated tissues, such as the vitreous cortex and internal limiting membrane (ILM), and the identification of activated glial cells are crucial for understanding the pathogenesis of LMHs. The vitreoretinal interface is composed of the vitreous cortex and the ILM. In young people, adherence of the vitreous to the retina involves the macular area and the optic nerve (sheet-like configuration) [52]. An adhesive layer of laminin, fibronectin, and type IV collagen is placed into the vitreoretinal interface and facilitates connections between the posterior vitreous and the ILM [53]. This adherence weakens over the years. PVD is due to dehiscence of the vitreoretinal interface as well as due to liquefaction of the vitreous body. These morphological changes induce the migration, differentiation, and proliferation of cells; vitreous cells (such as hyalocytes and fibroblasts), macroglial cells (such as astrocytes and Müller cells), and retinal pigment epithelial (RPE) cells have been implicated [54, 55].

Immunohistochemistry has been widely used to understand the distribution and localization of biomarkers: glial fibrillary acidic protein (GFAP), a marker of the intermediate filaments of glial cells; cellular retinaldehyde-binding protein, a marker of glial cells and RPE cells; neurofilament, a marker of retinal ganglion cells; α-smooth muscle actin (α-SMA), an antibody to intracellular actin filaments; CD45 and CD64, markers of hyalocytes; and CD68, a marker of macrophages/microglia [3, 7].

Immunohistochemical and microscopic studies have highlighted three important findings. Firstly, CD45, CD64, and GFAP were the most frequent cell markers found in both types of ERM, C ERM and A ERM, suggesting that hyalocytes and glial cells play an important pathogenic role [3, 7, 56–60]. Otherwise, cellular retinaldehyde-binding protein was positive only in a few samples, supporting the hypothesis that RPE cells are not implicated. The studies of CD68 antibodies and neurofilaments produced contrasting results [3, 7, 56–60].

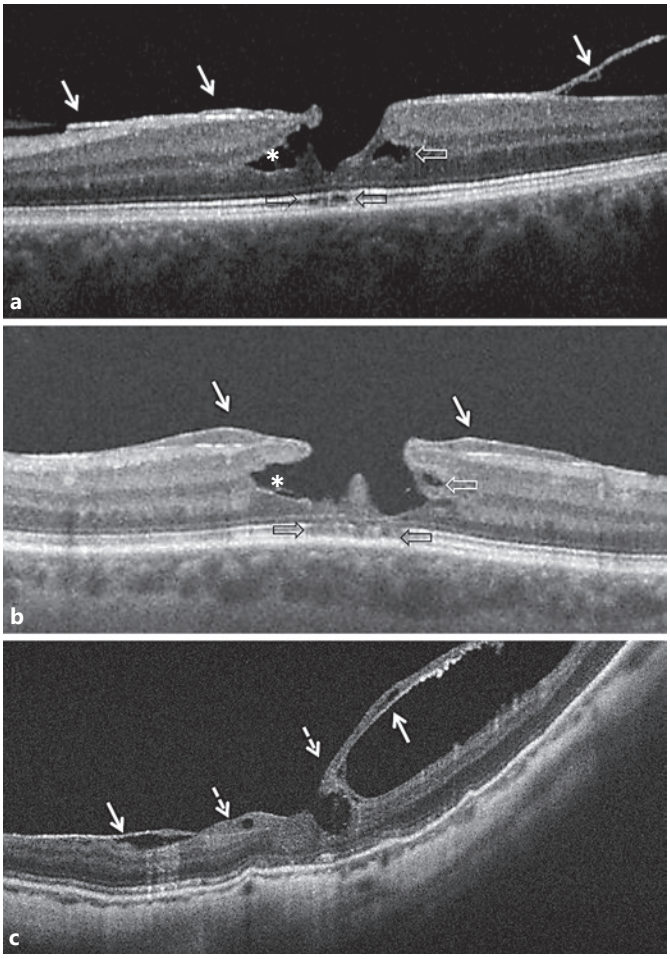


Fig. 1. Lamellar macular hole (LMH) with conventional epiretinal membrane (C ERM) and atypical ERM (A ERM). **a** LMH with C ERM. The ERM appears as a hyperreflective line (white arrows), intraretinal splitting (asterisk), intraretinal cysts (white empty arrow), and a defect of the outer retinal layers (black empty arrows). **b** LMH with A ERM. The ERM appears as a thickened membrane (white arrows), intraretinal splitting (asterisk), intraretinal cysts (white empty arrow), and a defect of the outer retinal layers (black empty arrows). **c** LMH with mixed ERM. The ERM has the characteristics of a C ERM (white arrows) and an A ERM (white dashed arrows).

The second finding regards the different staining of the two types of ERM with α-SMA antibody [25, 56]. This protein stained faintly positively or was absent in the A ERM, whereas it stained strongly positively in the C ERM. α-SMA is a protein that gives contractile properties to cells and tissues; thus, the perceptibly positive staining of the C ERM could explain the tractional properties of this membrane.

The last finding regards the collagen composition of the ERM. Native vitreous collagen (NVC) has been detected in both types of ERM. However, the A ERM is also composed of abundant clusters of compact fibrous long-spacing collagen (FLSC). FLSC is characterized by an irregular distribution of fibrils, which appear with a larger diameter than that of native vitreous collagen. FLSC is the result of the degradation of normal collagen fibrils. A remodeling process in the vitreous cortex, supported by persistence of adhesions of the posterior hyaloid and by the presence of abundant clusters of FLSC, has been hypothesized to play a role in the development of A ERM. In contrast to this theory, some authors have hypothesized that Müller cells are mainly responsible for A ERM pathogenesis, supported by the immunohistochemical GFAP positivity. The evidence that supports this theory is varied. First of all, A ERM is intimately connected to the underlying retinal tissue, and the only cells that span the entire retinal thickness are Müller cells. Moreover, Müller cells may proliferate and become hypertrophic, and thus be able to form an epiretinal tissue. Finally, Müller cells are implicated in tissue repair processes [3, 6, 11, 55, 56]. Other studies have raised doubts about the specificity of GFAP for identifying Müller cells. It has been shown that the CD45 and CD64 markers, specific for hyalocytes, are positive in colocalization with GFAP. The double positivity for specific antibodies against cells of a different nature could be due to the phagocytic activity of debris from Müller cells, by macrophages that acquire GFAP-positive receptors [5, 56–60]. Furthermore, the differentiation processes may cause a change in the receptors that distinguish them in physiological conditions. Glial cells lose expression of GFAP during differentiation in myofibroblast-like cells, with a consequent gain in α-SMA immunoreactivity [5, 56, 60–63].

The most recent theories were proposed by Son et al. [14] and Obana et al. [12]. Son et al. [14] detected the presence of RPE cells in surgically excised ERMs and isolated from ILMs [62], hypothesizing that RPE cells migrate through the intraretinal defect of an LMH and proliferate in the vitreoretinal interface [14]. Obana et al. [12] detected xanthophyll pigment in the composition of the A ERM, hypothesizing that this pigment is released by Müller cells. The absence of pigment inside the LMH, where Müller cells were missing, and its reappearance after restoration of the foveal depression (i.e., when Müller cells had restored the foveal architecture), suggests that the pigment was bound to these cells.

Diagnosis

Currently, OCT and fundus autofluorescence are the diagnostic tests that offer complete information about the tissue damage caused by an LMH. OCT is the gold standard for the diagnosis of LMH. It provides measurable and repeatable data on the anatomical damage to the specific retinal structures and allows following the development of this disorder. Fundus autofluorescence provides information regarding the integrity of the fovea via analysis of spontaneous tissue fluorescence. The blue autofluorescence stems from the lipofuscin present in the retinal pigment epithelium. In the macular area, it is attenuated by the presence of lutein pigment. Thinning or loss of tissue in the foveal area causes an increase in autofluorescence due to a reduction of the masking effect of lutein pigment [64] (Fig. 2).

Recent studies have shown the advantages of using en face SD-OCT. En face SD-OCT permits the observer to see the distribution of the ERM and the presence or absence of a contraction epicenter. Some authors have re-evaluated the dynamics of traction in LMH, highlighting how development of an intraretinal split in the LMH is not always due to a centrifugal tractional force on the fovea, but rather is a much more complex multivectorial process involving tractional forces from opposite directions [65, 66]. The ERM generates tangential traction lines that may not be seen on OCT. Microperimetry allows the observer to identify the stability of fixation and functional alterations before a symptomatic loss of visual acuity [67] (Fig. 2).

Clinical Manifestations

The symptoms of LMH are similar to those found in other vitreoretinal interface syndromes. They are characterized by decreased visual acuity, metamorphopsia, and perception of a central dark spot (central scotoma). Symptoms are often absent or blurred in the initial stages of LMH, but they may worsen over time with slow and

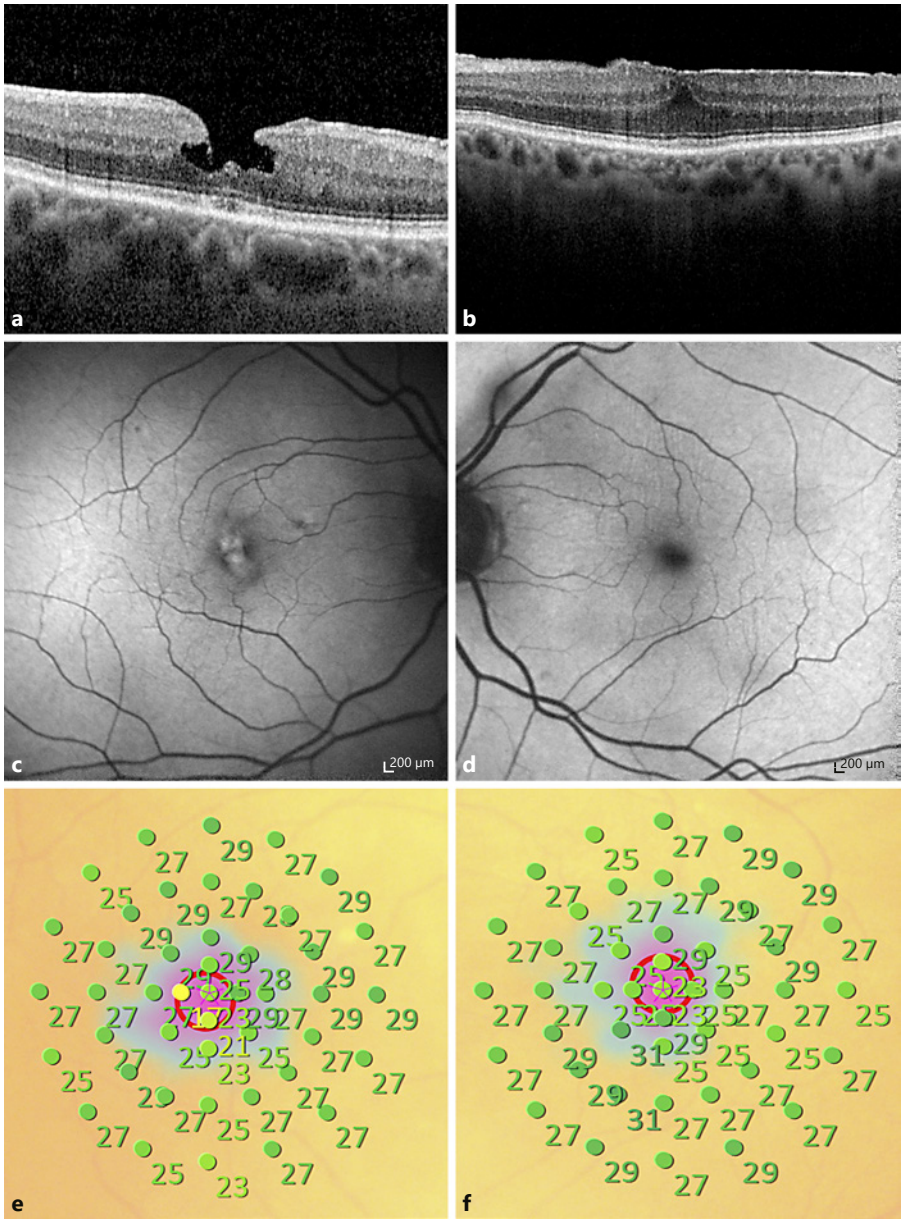


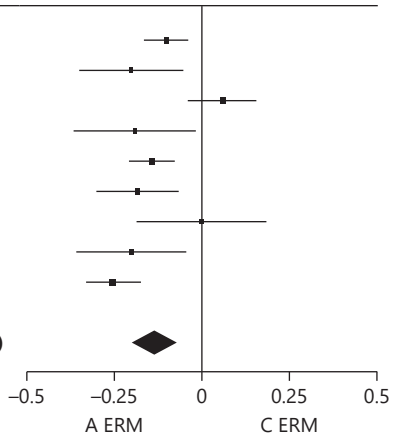
Fig. 2. Images from a patient affected by a lamellar macular hole (LMH) in the right eye and macular pucker (MP) in the left eye. Optic coherence tomography shows an LMH in the right eye (**a**) and MP in the left eye (**b**). Fundus autofluorescence (FAF) shows hyper-FAF in correspondence with the LMH in the right eye (**c**) and normal FAF in the left eye (**d**). Microperimetry shows functional damage to the right eye (2 scotomas in the foveal area; **e**) and no damage to the left eye (**f**).

gradual evolution [68]. Visual acuity may be correlated with ERM type: A ERM is associated with lower visual acuity than C ERM. Figure 3a shows the comparison of BCVA between LMH associated with C ERM and LMH associated with A ERM ($p < 0.0001$) [3, 4, 7–11, 22]. The difference in visual acuity may be explained by a greater involvement of outer intraretinal layers ($p = 0.0003$) and of the ellipsoid zone and external limiting membrane (ELM), and by a lower residual foveal thickness in LMH associated with A ERM than in LMH associated with C ERM ($p = 0.00001$) [3, 4, 8–10, 22] (Fig. 3b, c). The integrity of the ELM seems to influence visual acuity. It has been shown that a defect of the ELM with an intact photoreceptor layer is associated with visual acuity impairment [3, 5, 6–8] (Fig. 2). Most LMHs do not progress anatomically over a long period of time and are not associated with a significant decrease in visual acuity. Several studies followed the natural history of LMH, demonstrating in some cases a reduction in central foveal thickness and increased intraretinal splitting with evolution to FTMH [22, 40, 55, 68–70].

a Meta-analysis of BCVA (logMAR)

Study or subgroup	C ERM mean	C ERM SD	C ERM total	A ERM mean	A ERM SD	A ERM total	Weight, %	Mean difference IV, random, 95% CI
Bottoni et al. [4], 2013	0.44	0.1	24	0.54	0.08	10	14.6	–0.10 (–0.16, –0.04)
Coassin et al. [11], 2018	0.41	0.19	69	0.61	0.31	19	9.3	–0.20 (–0.35, –0.05)
Compera et al. [7], 2015	0.46	0.06	3	0.4	0.1	8	12.4	0.06 (–0.04, 0.16)
dell'Omo et al. [10], 2018	0.16	0.15	43	0.35	0.28	11	8.0	–0.19 (–0.36, –0.02)
Govetto et al. [9], 2016	0.13	0.12	43	0.27	0.2	59	14.7	–0.14 (–0.20, –0.08)
Pang et al. [22], 2014	0.331	0.312	83	0.512	0.39	62	11.0	–0.18 (–0.30, –0.06)
Parolini et al. [3], 2011	0.4	0.2	8	0.4	0.2	11	7.5	0.00 (–0.18, 0.18)
Schumann et al. [6], 2015	0.29	0.15	20	0.49	0.28	15	8.8	–0.20 (–0.36, –0.04)
Zampedri et al. [8], 2017	0.14	0.16	117	0.39	0.31	72	13.7	–0.25 (–0.33, –0.17)
Total (95% CI)			**410**			**267**	**100.0**	**–0.13 (–0.20, –0.17)**

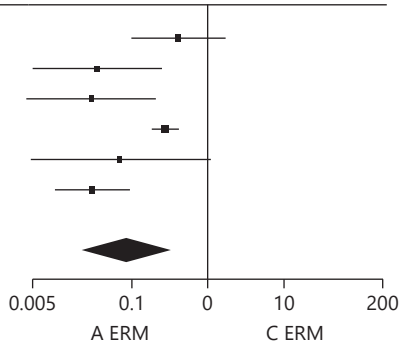
Heterogeneity: $\tau^2 = 0.01$; $\chi^2 = 29.71$, df = 8 ($p = 0.0002$), $I^2 = 73\%$
Test for overall effect: $Z = 3.92$ ($p < 0.0001$)



b Meta-analysis of the EZ-ELM (%)

Study or subgroup	C ERM events	C ERM total	A ERM events	A ERM total	Weight, %	Risk ratio M-H, random, 95% CI
Bottoni et al. [4], 2013	3	24	3	10	17.7	0.42 (0.10, 1.72)
dell'Omo et al. [10], 2018	1	43	27	41	15.0	0.04 (0.01, 0.25)
Govetto et al. [9], 2016	1	43	46	59	15.1	0.03 (0.00, 0.21)
Pang et al. [22], 2014	20	83	55	62	21.6	0.27 (0.18, 0.40)
Parolini et al. [3], 2011	0	8	9	11	11.6	0.07 (0.00, 1.05)
Zampedri et al. [8], 2017	3	117	61	72	19.1	0.03 (0.01, 0.09)
Total (95% CI)		**318**		**255**	**100.0**	**0.09 (0.02, 0.33)**
Total events	28		201			

Heterogeneity: $\tau^2 = 2.11$; $\chi^2 = 32.11$, df = 5 ($p < 0.00001$), $I^2 = 84\%$
Test for overall effect: $Z = 3.58$ ($p = 0.0003$)



c Meta-analysis of residual foveal thickness (μm)

Study or subgroup	C ERM mean	C ERM SD	C ERM total	A ERM mean	A ERM SD	A ERM total	Weight, %	Mean difference IV, random, 95% CI
Bottoni et al. [4], 2013	190	21	24	155	33	10	8.6	35.00 (12.89, 57.11)
dell'Omo et al. [10], 2018	196	36	43	163	48	41	12.7	33.00 (14.79, 51.21)
Govetto et al. [9], 2016	140.2	20.3	43	101.1	34	59	37.7	39.10 (28.51, 49.69)
Pang et al. [22], 2014	132.7	43	83	93.3	39	62	23.5	38.80 (35.39, 52.21)
Zampedri et al. [8], 2017	152.15	77.25	117	101.06	29.37	72	17.5	51.09 (35.54, 66.64)
Total (95% CI)			**310**			**244**	**100.0**	**39.99 (33.49, 46.49)**

Heterogeneity: $\tau^2 = 0.00$; $\chi^2 = 2.78$, df = 4 ($p = 0.60$), $I^2 = 0\%$
Test for overall effect: $Z = 12.06$ ($p < 0.00001$)

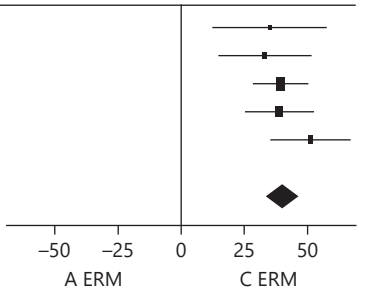


Fig. 3. Meta-analysis of the comparison of functional (**a**) and morphological (**b**, **c**) parameters between lamellar macular hole associated with C ERM and lamellar macular hole associated with A ERM (forest plot). A ERM, atypical epiretinal membrane; C ERM, conventional epiretinal membrane; BCVA, best corrected visual acuity; EZ, ellipsoid zone; ELM, external limiting membrane.

Table 1. Descriptive meta-analysis of surgical procedures, results, and postoperative complications in the treatment of LMH

Study [Ref.], year	*n*	ERM type, *n*	Surgery			Prone position	Visual improvement	Postoperative complications	
		C ERM A ERM mix = C + A ERM	gauge	peeling	tamponade	yes (days)	*sig	FTMH, *n* (%)	cataracts/ phakic eyes, *n*/*n* (%)
Hirakawa et al. [79], 2005	2	na	na	ERM + ILM	SF_6	yes (7)	*	0	0
Witkin et al. [2], 2006	4	na	na	ERM	SF_6 (20%)	na	no	2 (50)	0
Garretson et al. [71], 2008	27	na	20-25-23	ERM + ILM	yes; na tamponade type	yes (1–7)	*	1 (3.7)	8/(na)
Androudi et al. [72], 2009	20	na	na	ERM + ILM	C_3F_8	yes (na)	*	0	8/20 (40)
Parolini et al. [3], 2011	19	C ERM 6 A ERM 13	na	ERM + ILM	air	no	*	C ERM 0 A ERM 3 (15.8)	0
Casparis and Bovey [73], 2011	45	na	23	ERM + ILM	air/SF_6	yes (1)	*	0	17/32 (53)
Figueroa et al. [75], 2011	12	na	25	ERM + ILM	C_3F_8	yes (14)	*	3 (25)	3/9 (33)
Sato et al. [82], 2015	41	na	23	ERM + ILM	air (23) no (18)	yes (1–3)	*	0	0
Lai et al. [76], 2016	43	C ERM 24 A ERM 19	23	ERM + ILM	C_3F_8	7	*	0	0
Dutra Medeiros et al. [77], 2015	1	na	23	ERM	yes	yes (5)	no	1	0
dell'Omo et al. [10], 2018	26	C ERM 14 A ERM 4 mix 8	25	ERM + ILM	na	na	*	C ERM 0 A ERM 3 mix 0	0
Ko et al. [78], 2017	73	C ERM 58 A ERM 15	20-23-25	ERM + ILM	C_3F_8	yes (7)	*C ERM no A ERM	0	3/(na)
Choi et al. [80], 2018	22	C ERM 11 A ERM 11	23	ERM + ILM	air	na	*C ERM no A ERM	C ERM 0 A ERM 3 (27.7)	na
Coassin et al. [11], 2018	106	C ERM 69 A ERM 19 mix 18	23	ERM + ILM	air/SF_6/C_3F_8	yes (3–7)	*C ERM no A ERM *mix	C ERM 0 A ERM 3 (4.3) mix 0	30

LMH, lamellar macular hole; A ERM, atypical epiretinal membrane; C ERM, conventional epiretinal membrane; ILM, internal limiting membrane; SF_6, sulfur hexafluoride; C_3F_8, perfluoropropane; FTMH, full-thickness macular hole; na, not available; *sig, significant improvement.

Surgical Treatment of LMH

Surgical treatment of LMH is indicated in patients with progressive visual loss associated with metamorphopsia and is also based on the risk-to-benefit ratio. The current approach in the treatment of LMH is pars plana vitrectomy. The rationale is to release vitreomacular adhesions by removing the ERM and ILM, facilitating re-establishment of the regular foveal profile. Some surgeons use temporary intravitreal tamponade, such as air or gas, and instruct the patient to maintain a prone position postoperatively [71–74, 76–80]. Others have demonstrated that intravitreal tamponade and postoperative prone positioning are not crucial for the surgical success [3, 75, 81, 82] (Table 1). Although the natural evolution of LMH appears to be slow, in almost all studies a statistically significant improvement in visual acuity after surgery has been observed.

Surgery, however, is not without risk. Development of cataract is the most frequent postoperative complication. Another one, more serious, is the development of an FTMH (Table 1). The presence of an A ERM is a risk factor for developing an FTMH. Most of the patients treated underwent the standard surgical procedure, in other words, removal of the ERM and ILM from the foveal area including the LMH. Removal of the ERM and ILM has been shown to induce closure of the LMH. On the other hand, surgical peeling may cause iatrogenic damage to Müller cells [83] with consequent alteration of the retinal structure and possible evolution to FTMH. Peeling of an A ERM requires several and more aggressive surgical maneuvers than those required to peel a C ERM [3, 10, 11, 55, 63, 84, 85]. Furthermore, the composition of A ERMs is still unclear, leaving doubts about the real benefit from its complete removal.

Shiraga et al. [84] have recently proposed an alternative surgical technique for treatment of LMH with A ERM, avoiding removal of the ERM and ILM in the LMH. The rationale of this technique is similar to that of the fovea-sparing ILM peeling technique for the treatment of myopic tractional maculopathy, proposed by Shimada et al. [85], and the inverted ILM flap technique for the treatment of FTMH, proposed by Michaleswka et al. [86]. With this technique there is a lower risk of iatrogenic damage, avoiding peeling of the ERM on the fovea, and the residual ILM around the fovea creates a support for the proliferation of Müller cells and the consequent release of growth factors that induce closure of the FTMH.

Conclusions

The current knowledge about LMH is still partly limited regarding its pathogenesis and treatment. There are two distinct clinical entities of LMH according to the type of ERM associated with it, C ERM and A ERM. These two types of ERM differ in their cell and collagen composition. LMHs with an A ERM are more compromised than LMHs with a C ERM, both functionally and morphologically. LMH is not a stable condition, but it tends to worsen over time. The standard surgical procedure yields excellent anatomical and functional outcomes, but it is not without risks, such as FTMH development. Further studies comparing the standard surgical procedure with newer ones, such as fovea sparing or inverted flap, are needed in order to establish the best therapeutic strategy.

Statement of Ethics

The authors have no ethical conflicts to disclose.

Disclosure Statement

The authors have no conflicts of interest to declare.

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